BRC
ANALYZING HOW INJURIES ARE CAUSED™

July 11, 2018

Mr. Lane R. Neal
Durbin, Larimore & Bialick
920 N. Harvey
Oklahoma City, OK 73102

Re: **Stiebens, Christopher vs. Stauffer, Leon**

Dear Mr. Neal:

Per your request, I am submitting this report regarding my opinions to date in the above referenced matter. The purpose of this report is to set forth my general qualifications and opinions to date of the injury mechanisms and injury causation involved in this incident based upon a biomechanical assessment of the incident scenario.

My education includes degrees in both engineering and medicine. I received a Bachelor of Science degree (Electrical Engineering) from Illinois Institute of Technology in 1987. In pursuit of that degree, I studied the sciences, including physics, chemistry, mathematics, metallurgy, statics, dynamics, and thermodynamics. I have a Bachelor of Science degree in Nursing (summa cum laude). Studies included all aspects of nursing care, including emergency nursing. I attended medical school at Midwestern University, and graduated in the top ten percent of my class. During the first two years of medical school, in addition to clinical medicine, I studied anatomy in the cadaver lab, physiology, biochemistry, psychology, and pathology. I completed core rotations in subjects such as internal medicine, pediatrics, cardiology, and surgery. There was also ample time for electives, and my choices included trauma and medical intensive care, emergency medicine, neurology, pathology in the county coroner's office, and radiology.

My professional career includes working at Ford Motor Company for six years as a test engineer and a fuel systems development engineer. During my training program at Ford, I worked on cars, light trucks, and heavy trucks. I was involved in exhaust and evaporative emissions testing, durability testing with stress and strain measurements on various structural and body components, and heavy truck performance and durability testing. After my training program, I was recruited by the truck division, and worked as a fuel systems development engineer. I was responsible for fuel system components in all current and past F-series light trucks and Econoline vans. I resolved assembly plant, customer and dealership concerns throughout North and Central America. I

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PLAINTIFFS' EXHIBIT 2

coordinated a safety campaign involving the recall of 1.5 million trucks to correct a fuel system concern.

During my career at Ford, I sought training as an emergency medical technician, and became a nationally registered EMT. I worked part time on weekends providing emergency care to the sick and injured. I also volunteered for my local fire department as a fire fighter/EMT, where I received training and experience with firefighting and vehicle extrication procedures to augment my patient care activities. Since 2016, I have been certified as a Fire/Arson Investigator.

During my eight years as a registered nurse, I worked in rural, reservation, and urban emergency departments. I achieved certification in emergency nursing. I pursued additional training in forensic nursing, and acted as my department's sexual assault examiner and domestic violence advocate.

I am board certified in emergency medicine after completing an emergency medicine residency in 2006 at the University of Kentucky. The vast majority of the three year program was spent caring for patients in the emergency department, but I also had rotations in trauma and surgical intensive care, pediatrics and pediatric intensive care, internal medicine, obstetrics, orthopedic surgery, air medical transport, and EMRS. During the last two years of residency, I worked in several rural emergency departments as the sole physician. I was also part of the team of physicians who provided emergency medical care at Kentucky Speedway. As my academic project, I co-authored a book chapter on the care of trauma patients.

After residency, I worked for six years at a rural emergency department in Wyoming. There were few resources or specialists, and I cared for all patients with very little support. I acted as medical director for the trauma service, evaluating and improving the care provided for all serious trauma patients seen in the ER. Additionally, I worked part time at a rural Indian Health Service emergency department in Montana, an urban Indian Health Service emergency department in Phoenix, two other rural Wyoming ERs, and a larger community Wyoming ER. Since 2008, I have been volunteering my medical expertise at clinics for the uninsured. Upon joining BRC, I continued working part time as an emergency physician. Since 2009, I have been volunteering as an examiner for the American Academy of Emergency Medicine Oral Board Review Course. I was recently appointed as an Assistant Clinical Professor at University of the Incarnate Word medical school.

Over the course of my more than twenty year career as an emergency provider, I have directly cared for thousands of trauma patients with neurological, orthopedic, cardiopulmonary, and abdominal injuries, some fatal, some life and limb-threatening, and some minor.

I am currently licensed to practice medicine in Texas, Wyoming and Montana. I am a Fellow of the American College of Emergency Physicians and the American Academy of Emergency Medicine. I hold certifications in Trauma, Cardiac, Burn, and Pediatric Life Support.

I am currently employed as a consultant in the field of biomechanics. In addition to my training and experience as a physician and automotive engineer, I have been trained in accident reconstruction and am certified by ACTAR. I am involved in current research efforts involving the accelerations experienced by people in everyday activities. I have co-authored papers regarding whole body accelerations, crash reconstruction, lumbar accelerations in everyday activities, head accelerations in low speed rear end collisions, and the risk of injury in low speed rear end impacts.

My professional background is documented in the attached copy of my curriculum vitae. My testifying history is documented in the attached document. My time is billed by Biodynamic Research Corporation at $445 per hour.

During the course of my study of this matter I have had access to the following items:

- Oklahoma Traffic Collision Report;
- Repair Estimate for 1997 Saturn SL1;
- Repair Estimate for 2011 Honda CRV;
- Various Pleadings and Discovery Responses;
- Depositions of:
  Chad Johnson
  Mary Stiebens
  Christopher Stiebens;
- Color Photographs of:
  3 Color Photographs of 2011 Honda CRV
  2 Color Photographs of Accident Scene
  Color Photographs from Facebook & Instagram for Christopher Stiebens
  1 Color Photograph of Accident Scene
  3 Color Photographs of 2011 Honda CRV
  10 Color Photographs of 1997 Saturn SL1;
- DVD's, CD's, Flash Drives of Downloads Containing Legal Documents; Medical Records, Repair Estimates, Photographs, and Imaging Studies;
- Medical Records for Christopher Stiebens from:
  Toy Chiropractic Clinic – James W. Toy, B.A., D.C.
  Chandler Fire/EMS
  Comanche County Memorial Hospital
  Ferrara Chiropractic – Anthony Ferrara, D.C.
  Fountain Park Family Physicians – Kim King, D.O.
  Dr. Dawn's Chiropractic Center, PLLC – Dawn Hacker, D.C., DACBSP
  Oklahoma Pain Management – Scott A. Mitchell, D.O.
  Neuroscience Specialists – Michael R. Hahn, II, M.D. and Fadi F. Nasr, M.D.
  Oklahoma Spine Hospital
  Oklahoma Diagnostic Imaging
  Mercy Hospital Oklahoma City
  Mercy Clinic Pain Management – Andrea Fraley, M.D.

Lawton Community Health Center – Moncy Varkey, D.O.
IME – Arcadia Medical Center – Stephen B. Conner, M.D.
Anderson Pharmacy; and

- Medical Records for Mary Stiebens from:
  Memorial Medical Group – Renato M. Caballero, M.D.
  Toy Chiropractic Clinic
  Comanche County Memorial Hospital
  Ferrara Chiropractic
  Fountain Park Family Physicians – Kim King, D.O.
  Community Hospital
  Oklahoma Interventional Pain Management – Scott Mitchell, D.O.
  IME – Arcadia Medical Group / Stephen B. Conner, M.D.

I have arrived at the following opinions, based upon a reasonable degree of medical and scientific certainty:

Incident

On June 19, 2014, Mrs. Mary Stiebens, age 44, was the restrained driver of a 2011 Honda CR-V which was traveling eastbound on I-44 in Lincoln County, Oklahoma. Mr. Christopher Stiebens, age 44, was the restrained right front passenger. According to the police report, a 1997 Saturn SL1 was traveling behind the Stiebens' Honda, and a 2009 Volvo tractor trailer was behind the Saturn. The Honda and the Saturn were stopped for congested traffic, and the Volvo struck the Saturn from behind, pushing the Saturn into the rear of the Stiebens' Honda. Mrs. Mary Stiebens was not injured. Mr. Christopher Stiebens had a possible injury, noting he had been in an accident previously, and may have reinjured his back. The Honda was driven from the scene.

In her May 24, 2017, deposition, Mrs. Mary Stiebens testified that she and her husband still owned the subject Honda, and had driven it to the deposition. She was restrained at the time of the collision, and saw the collision between the Volvo and Saturn in her rearview mirror. She had slowed her vehicle for traffic, and was going less than 5 mph ("maybe going one" mph) at the time of impact by the Saturn. Her vehicle did not impact the vehicle ahead of her. Her injuries included her neck, upper back, and headaches.

In his May 24, 2017, deposition, Mr. Christopher Stiebens testified that he was restrained at the time of the collision. They had slowed almost to a stop, "barely rolling forward". He was facing forward with his arms on the armrests. He had no warning of the impending impact. His wife drove the vehicle from the scene. His injuries included his neck, mid back, lower back, left arm, fogginess of thinking, and tinnitus. He had undergone lumbar and cervical surgery.

Medical

According to the available records, Mrs. Mary Stiebens had a past history of smoking, morbid obesity status post-lap band surgery, prior motor vehicle collision, osteoporosis, hypertension, acid reflux, and arthralgia. She first sought care the day after the subject incident at a chiropractic office, with complaints of headaches, neck pain, low back pain, *right* leg pain, and blocked left ear. She was noted to have been belted with the head restraint above her head, and unaware of the impending impact. She was noted to have injured her head, but had no loss of consciousness. One week after the collision, she was seen in the ER with complaints of headache, facial pain, neck pain, low back pain, and *left* leg pain. She denied head injury and loss of consciousness. On exam, she was noted to be in no distress and looking around. Her head was atraumatic, her face was non-tender, and her neck was supple and non-tender. Her neurologic exam was normal. Imaging of her cervical spine demonstrated degenerative changes, and no sign of acute injury. Subsequent cervical spine MRI demonstrated degenerative changes, but no sign of acute injury. In follow-up with a different chiropractor, she described having been aware of the impending impact. Her complaints included head, neck, and low back pain. She had also developed numbness of her entire left arm. In follow-up with family practice, she again described in detail having been aware of the impending impact. She further noted, "my neck was thrown forward and back as I braced for impact." Another cervical spine MRI again demonstrated degenerative changes, and no acute injury. By December 2014, she had developed trouble focusing. By April 2015, she had developed weakness in her left arm and hand. She underwent cervical epidural steroid injections, facet blocks, and radio frequency ablation with improvement.

According to the available records, Mr. Christopher Stiebens had a past history of smoking, gunshot wound to his left leg, three prior motor vehicle collisions, on-going neck pain since the early 1990s, on-going low back pain since the early 2000s, headaches, bilateral shoulder pain, upper/mid back pain, bilateral hip pain, pain in all four extremities, rib pain, and left leg tingling. He was seen by the chiropractor one month prior to the subject incident with mid and low back pain, bilateral hip pain, and joint subluxations in his cervical, thoracic, and lumbar spine. On the date of incident, EMS responded to the scene, and noted Mr. Stiebens to be "without injuries". Trauma assessment was noted to be negative, and he was noted to have a Glasgow Coma Scale (GCS) of 15. He declined transport. The next day, he presented to a chiropractor with complaints of neck, entire back, and left arm pain. He was noted to have been belted with the head restraint above his head. There was no loss of consciousness. Examination revealed better hearing in his right ear than left. A few days later he had developed numbness in his left hand, as well as pain in his left leg. A week post-incident, he presented to the ER with complaints of neck pain radiating to his arm. He had by then developed left arm weakness. On exam, his head was noted to be atraumatic, and he had a GCS of 15. His neck was supple and non-tender. Cervical and thoracic spine x-rays demonstrated no acute injury. Subsequent cervical and thoracic MRIs demonstrated degenerative changes, and no acute injury. Chiropractic follow-up noted that he had been disoriented, dazed, and confused during and immediately following the collision. By August 2014, he had developed buzzing and ringing in his ears, as well as head pain. By July, he had developed left facial pain. In follow-up with family practice, he reported no loss of consciousness, but having been dazed after the incident. In follow-up with pain management in October 2014, he denied any

neck or back pain prior to the subject incident. He underwent cervical and thoracic epidural steroid injections. By October 2014, he had developed numbness in bilateral toes. By February 2015, in follow-up with neurosurgery, he had developed bilateral hand numbness. Exam revealed normal strength in all four extremities. Lower thoracic spinal surgery was planned. Repeat thoracic spine MRI again demonstrated degenerative changes, but "near complete resolution" of central disc protrusion and cord compression. Surgery was cancelled. He underwent medial branch blocks. A third thoracic MRI and a lumbar MRI demonstrated degenerative changes. He underwent lumbar epidural steroid injections. Neurosurgery follow-up notes indicated that he had a "pristine" lumbar spine, but if he had a pars defect, that may explain his symptoms. CT scan of his lumbar spine demonstrated bilateral L5 pars defects with no evidence of spondylolisthesis, degenerative changes, and no acute fracture. He underwent L5 laminectomy, reduction of spondylolisthesis, and fusion. He later underwent right hip injections for osteoarthritis. In neurosurgery follow-up he was noted to have had a cervical discogram with nerve root block, and cervical surgery was planned.

No further records were available at the time of this report.

Radiology

On June 20, 2018, I reviewed Mr. Stiebens' imaging studies with Dr. Cynthia Day, BRC's board certified radiologist. x-rays of his cervical, thoracic, and lumbar spine performed in 2009, over five years prior to the subject incident, demonstrated degenerative changes, as well as bilateral L5 pars defects and resulting anterolisthesis of L5 on S1. Spinal x-rays performed shortly after the date of incident demonstrated no change from Pre-incident films. No evidence of acute injury was seen on pre- or post-incident spinal x-rays. Subsequent MRIs of his spine demonstrated degenerative changes, and no evidence of acute injury. Further detail may be found in Dr. Day's report.

No imaging studies for Mrs. Mary Stiebens were available at the time of this report.

Impact Analysis

According to the impact analysis of Mr. Enrique Bonugli, the Stiebens' Honda experienced a change in speed or delta-V of 1.5 – 3.2 mph, resulting in a peak acceleration of 0.5 – 2.4 g. The principal direction of force (PDOF) was 6 o'clock.

Exemplar Surrogate Demonstration

I conducted exemplar surrogate demonstrations at BRC on July 6 and 9, 2018, using a closely matched exemplar vehicle and human surrogates matched to Mr. and Mrs. Stiebens for standing stature and weight. The female surrogate was positioned in the driver's seat and was asked to adjust the seat, steering wheel, and seatbelt D-ring to match her usual safe driving practices. She then donned the lap/shoulder belt. The male surrogate was positioned in the right front passenger seat and was asked to adjust the seat, and seatbelt D-ring to match his usual safe riding practices.

He then donned the lap/shoulder belt, and positioned his arms on the armrests. Occupant kinematics in the subject impact were demonstrated. In upright posture, the driver surrogate's head was less than 3 inches from the head restraint, and the passenger surrogate's head was in contact with the head restraint.

Vehicle Dynamics and Occupant Kinematics

Occupant kinematics for Mr. and Mrs. Stiebens during the subject incident were determined using the reviewed materials, the impact analysis, the radiology review, the exemplar surrogate demonstration, current literature, and the laws of physics. Newton's first law of motion states an object in motion will remain in motion at a constant speed and direction until acted upon by a force (Young). As the front of the Saturn contacted the rear of the Stiebens' Honda, and the Honda was pushed forward, Mr. and Mrs. Stiebens would remain at their pre-impact speed and direction. In the frame of reference of the vehicle's interior, they would appear to move backward (Fricke, McConnell 1993, McConnell 1995). Their heads would move slightly backward, and may contact the head restraints, and their thoracic and lumbar spines may extend slightly. On rebound, they may move forward minimally, and their spines may flex somewhat. Rearward movement and spinal extension were limited by the seatbacks and head restraints, and forward movement and spinal flexion were limited by the locked (Anderson, Furbish) seatbelts. Net spinal motion was within physiologic limits (Banks, McConnell 1993, McConnell 1995, Szabo 1994).

Injury Causation

Injury potential for Mr. and Mrs. Stiebens during the subject incident was determined using the reviewed materials, the impact analysis, the radiology review, the exemplar surrogate demonstration, current literature, and the laws of physics. The subject acceleration of 0.5 – 2.4 g was in the realm of everyday activities (e.g., dancing, riding an elevator), and would not be expected to cause injury (Figure 1 & Figure 2). Since Mr. and Mrs. Stiebens likely performed many of the activities shown on a regular basis without injury, there was no mechanism for them to be injured in an event with similar accelerations. Accordingly, no evidence of acute injury was demonstrated in their medical records, including imaging studies.

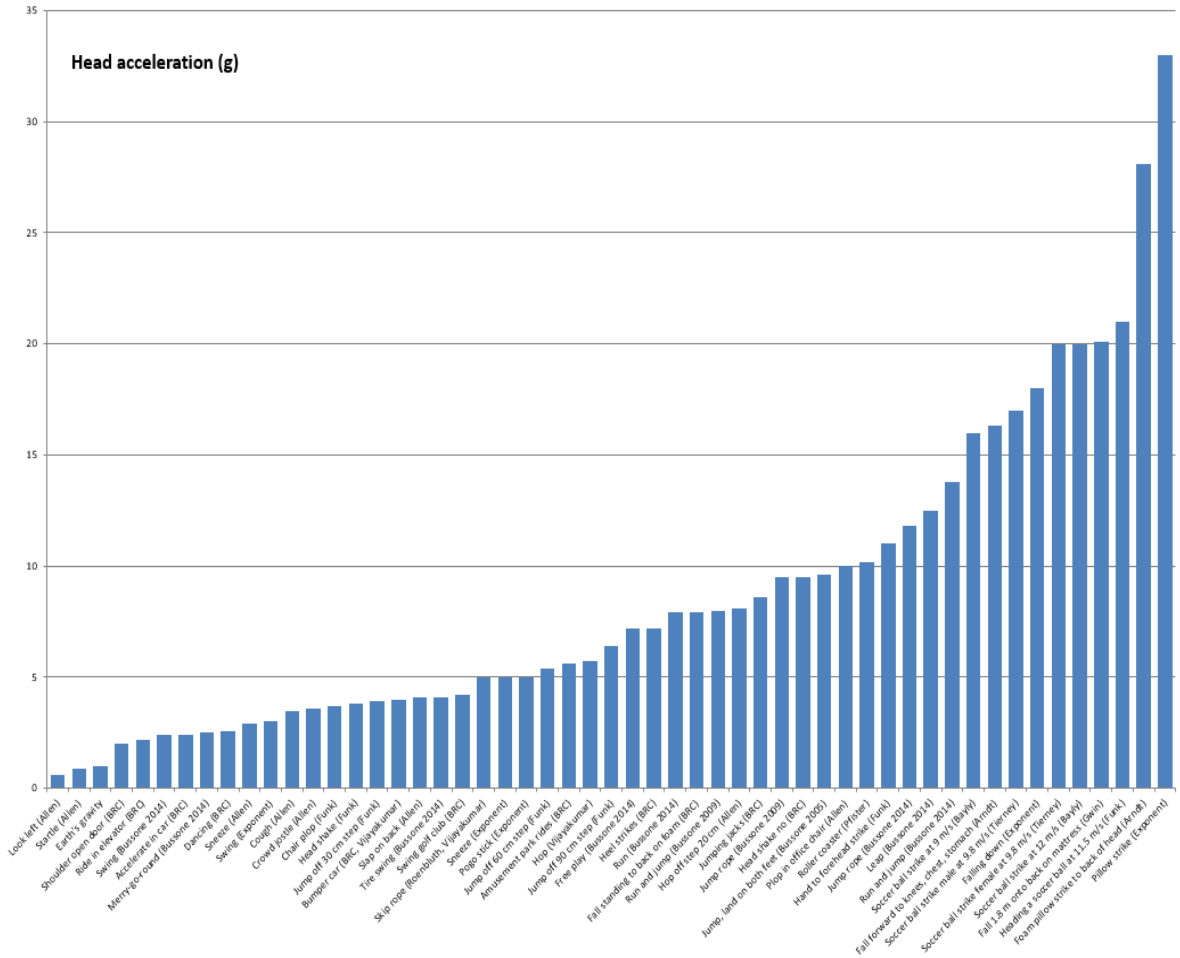

Figure 1. Head accelerations in everyday activities

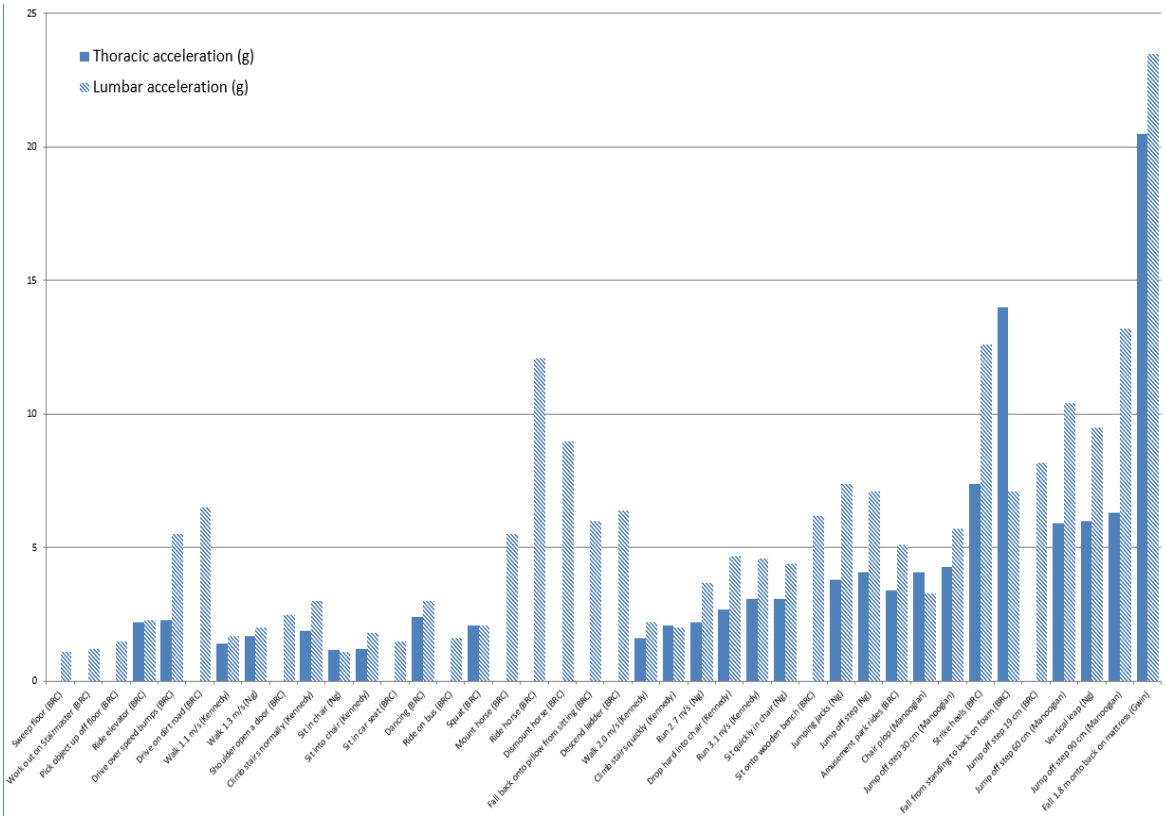

Figure 2. Thoracic and lumbar accelerations in everyday activities

The subject delta-V of 1.5 – 3.2 mph placed the subject incident in the category of "low" or "very low" speed (Lee, Viano 2007, Viano 2008a). Accordingly, the risk of serious and severe injuries in rear-end collisions with delta-Vs up to 30 mph [up to 9 times that of the Stiebens impact] is less than 1% (Viano 2008a). See Figure 3 below.

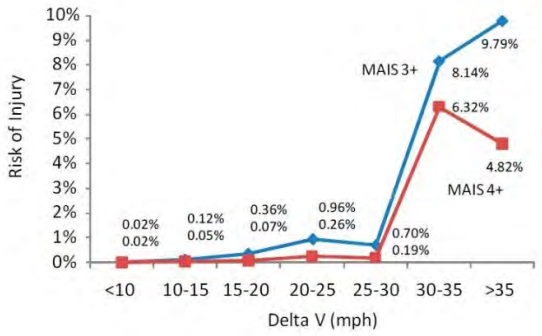

Figure 3. The risk of serious and severe injuries in rear-end collisions

A large amount of voluntary testing has been performed to evaluate the injury potential of low-speed rear impacts at this severity and higher. Within the voluntary testing performed at severities similar to the subject event the majority of the subjects did not report symptoms (Anderson, Bailey, Brault, Castro 1997, Croft, Eichberger, Fugger, Goodwin, Linder, McConnell 1993, McConnell 1995, Nielsen, Ono 1998, Rosenbluth, Siegmund 1994, Szabo 1994, Szabo 1996, Van Den Kroonenberg, Watanabe, Welcher, West). Due to the startling nature of these events, transient symptoms have been reported which resolve spontaneously (Castro 1997, Castro 2001). When symptoms of neck pain or decreased range of motion were reported they typically resolved within two weeks. Therefore, long-term symptoms related to the cervical spine are not expected to occur as a result of an event at this severity. These volunteer studies were performed within the United States and internationally after approval by an ethics review board which points to the broad, scientific acceptance that these events are benign. Szabo and Welcher (1996) compiled 284 volunteer crash tests which included collisions in the delta-V range experienced by Mr. and Mrs. Stiebens in the subject incident. None of the volunteers had symptoms lasting longer than 7 days (Figure 4).

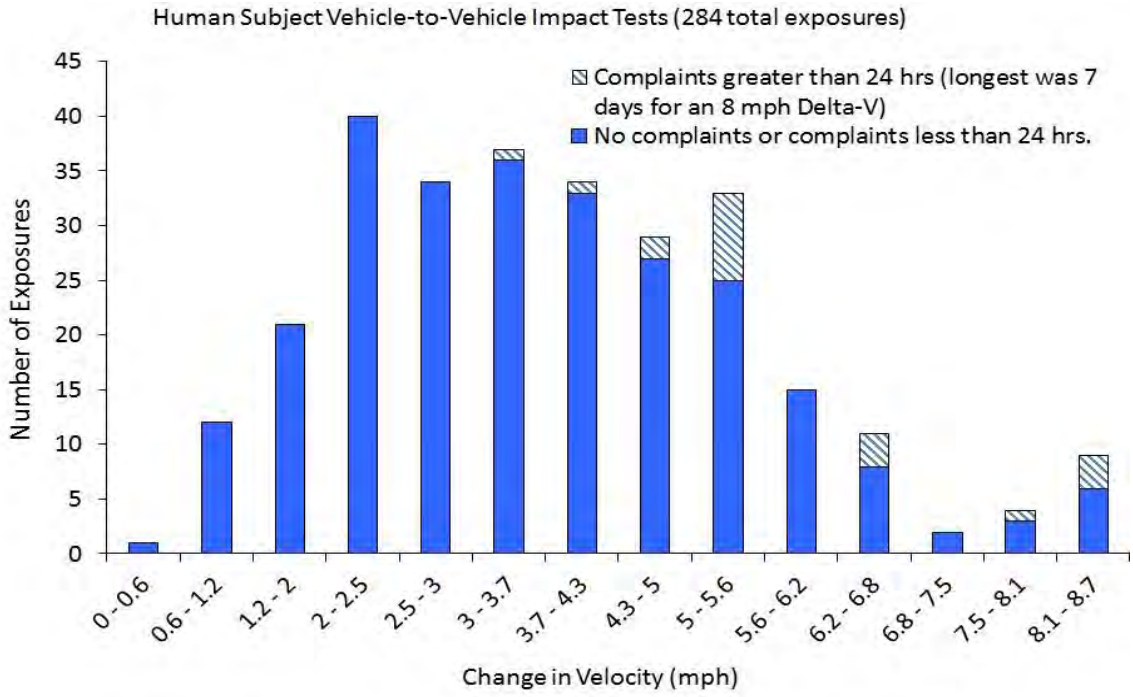

Figure 4. Symptoms in volunteer crash tests (Szabo 1996)

A recent paper (Cormier) performed an up to date metanalysis of 51 volunteer studies which produced a dataset of 1,984 volunteer impacts. Additionally, the authors analyzed a separate dataset of 515,601 weighted occupants in real-world rear collisions. They concluded that the prevalence of symptoms among volunteers matched that of occupants in real world crashes. They further found that rear impacts up to 25 mph delta-V did not result in any risk of AIS 2+ (moderate or greater) injury. The risk of injury at the delta-V the Stiebenses experienced was negligible.

Brain injury potential

Head accelerations are correlated to the amount of brain tissue strain that occurs with an application of force to the head. The risk of head injury, including concussion, is related to the level of acceleration experienced by the head in an event (Duma 2005, Funk 2007a, Pellman Vorst). Crash tests have determined that occupants' head accelerations are approximately 1 – 4 times the vehicle acceleration in rear-end collisions (Anderson, Fugger, Goodwin, Ono 1997, Ono 1999, Rosenbluth, Siegmund 1997, Szabo 1994, Szabo 1996, Van Den Kroonenberg, Welcher, West). However, in 90 crashes with vehicle accelerations up to 10 g, the average multiplier was 2. Two recent papers showed head accelerations in a more modern vehicle were 1 – 3.2 times the vehicle acceleration (Guzman, Scott). This means that the Stiebenses' peak head accelerations were between approximately 0.5 and 7.7 g. Various activities result in similar or greater head accelerations. Plopping into a chair can generate head accelerations as high as 10 g (Allen, Funk 2007b). Striking one's head with the heel of their hand can generate peak head accelerations as high as 18 g (Schewchenko). Heading a soccer ball at a moderate speed can result in head accelerations up to 30 g (Funk 2009, Naunheim, Schewchenko), as can pillow fights (Arndt).

The likelihood of a concussion, also known as a minor traumatic brain injury (mTBI), increases as impact severity and head acceleration increase. Older studies have proposed linear head acceleration thresholds for an mTBI of 42 g (Stapp) and 80 g (Patrick). A paper by Mertz gave a brief review of peak head accelerations and HIC (head injury criterion) and provided current injury assessment reference values for various anthropomorphic test devices. A recent study (Funk 2012) reported the recording of 37,128 head impacts and documented that the linear head accelerations ranged from 10 to 260 g. There were four mTBIs at an average head acceleration of 145 ± 35 g. An earlier analysis of this data showed that the 10% risk of an mTBI is a linear head acceleration of 160 g or a HIC of 400 (Funk 2007a). Another recent study (Rowson 2011) also provided risk curves for an mTBI (Figure 5). In the laboratory, traumatic brain injuries have been induced with pure rotation (Gennarelli). In real life, head impacts result in both translational and rotational accelerations. It has been shown that as rotational or translational accelerations increase, so does the other. Rowson (2013) showed that the best predictor of concussion was translational acceleration alone as opposed to rotational acceleration alone or a combination of translational and rotational accelerations. For the Stiebenses, their risk of an mTBI from the subject event was essentially zero.

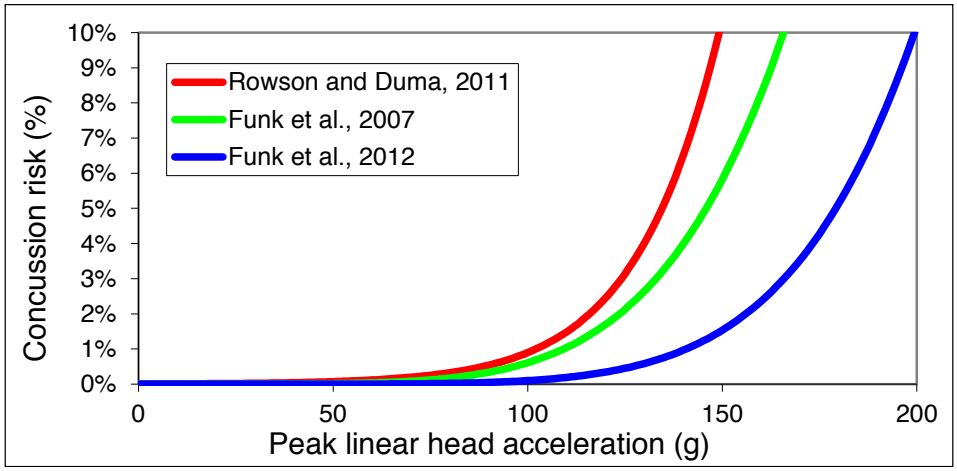

Figure 5. Risk of mTBI vs. peak head acceleration

A head injury producing loss or alteration of consciousness (with a LOC of less than 30 minutes and post-traumatic amnesia of less than 24 hours) associated with a Glasgow Coma Scale score between 13 and 15 after 30 minutes post-injury is generally felt to be compatible with an mTBI (in the setting of a normal CT or MRI brain scan). Various mTBI scoring systems exist. Most systems utilize three grades including The American Academy of Neurology. A Grade 1 concussion involves confusion with no LOC and associated symptoms lasting less than 15 minutes. A Grade 2 concussion also involves confusion with no LOC but associated symptoms lasting longer than 15 minutes. A Grade 3 concussion involves LOC. While some classification systems define the mildest form of traumatic brain injury as being short-lasting confusion, all classification systems require that impaired cerebral functioning be present. The American Academy of Neurology recently updated their guideline regarding concussion in sports (Giza). Two checklists have been validated to determine the likelihood of a concussion; according to their testimony regarding the time of incident, as well as their presentation to medical providers, neither Mr. nor Mrs. Stiebens had a concussion.

In my experience dealing with hundreds of patients immediately after an mTBI (with a significant percentage seen within minutes), short term memory deficits are extremely common and noted by repeated questioning (perseveration) about a specific topic. The patient is unable to remember asking a question or being given an answer. mTBI patients are almost always amnestic for the event that caused their concussion and amnesia may be considered a necessary condition to diagnose someone with an mTBI. The severity of the concussion is marked by the amount of retrograde and anterograde amnesia.

Iverson et al. has shown that post-concussive symptoms (PCS) are not unique to traumatic brain injured patients, but are commonly found in healthy individuals and are highly correlated with depressive symptoms. Also, Meares et al. has shown that PCS are seen in trauma patients equally, whether or not they sustained an mTBI, indicating that PCS are not specific for an mTBI. Carroll et al. has shown that there are no objectively measured mTBI-attributable cognitive deficits beyond

one to three months post-injury in the majority of cases. Contrary to the natural history of mTBI symptoms, individuals in litigation were associated with stable or worsening of cognitive functioning over time (Belanger). Mooney et al. showed that in cases of poor recovery after mTBI where compensation or litigation may be a factor, most of the variants in recovery seem to be explained by depression, pain and symptom invalidity rather than the injury variables themselves.

Spinal injury potential

Neck loads in rear-end collisions with delta-Vs of 15 – 27 mph (up to 8 times higher than the delta-V the Stiebenses experienced) have been analyzed by Viano et al. (2008b). The average cervical spine tensile load was 228 lbs. and the average compressive load was 98 lbs. Extension and flexion moments averaged 13.75 and 15.4 ft-lbs., respectively. Recommended injury assessment reference values (IARVs) are: tension – 937 lbs.; compression – 899 lbs.; extension moment – 100 ft-lbs.; flexion moment – 229 ft-lb (Eppinger). IARVs are used in industry guidelines and government regulations to define a level of biomechanical force that poses a low risk of injury (for the neck this risk is approximately 20% for a serious or greater injury). As shown in Figure 6, the Stiebenses were exposed to forces that placed them at an extremely low risk for a serious neck injury.

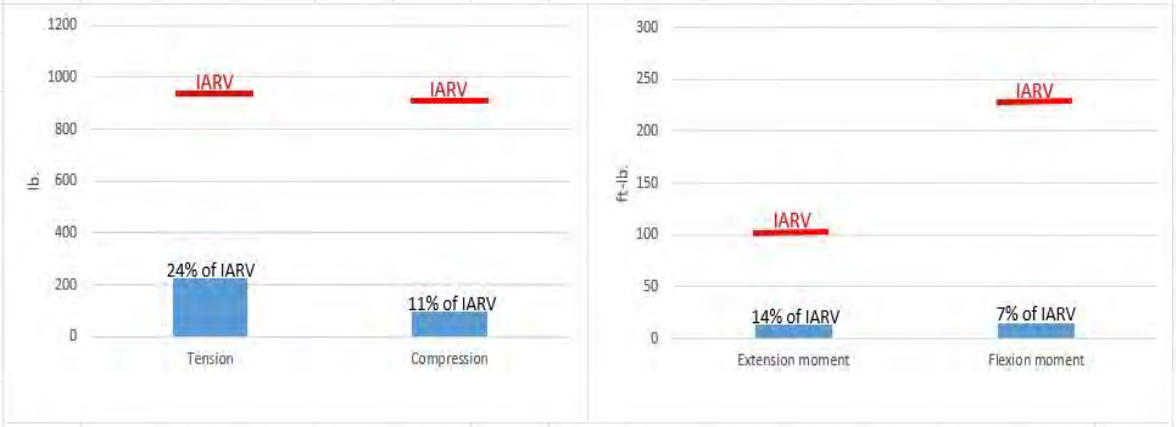

Figure 6. Neck loads in crash tests compared to IARVs

Similarly, Gates measured lumbar loads in rear end collisions with delta-Vs of 5-15 mph, also higher than the delta-V Mr. and Mrs. Stiebens experienced. As shown in Figure 7, peak compressive loads ranged from 13 to 196 lb-f, well below the failure loads seen by Adams (1982) and Yoganandan (1988). The Stiebenses were exposed to forces that placed them at an extremely low risk for a serious back injury.

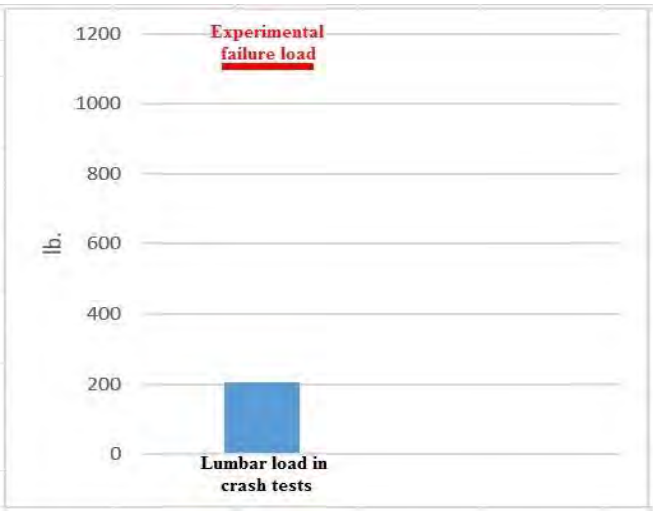

Figure 7. Peak compressive loads in crash tests compared to lumbar failure loads

Both Mr. and Mrs. Stiebens were noted to have disc abnormalities on imaging studies. Isolated bulging or herniation of the intervertebral discs is a degenerative process, not an acute injury (King, Lam, Yang). Many researchers have demonstrated this experimentally. Roaf compressed cadaveric spinal segments to the point that the vertebral bodies fractured, and no disc herniation occurred. Duma and colleagues (2008) performed similar testing which resulted in vertebral body fractures, ligamentous injuries, and disc "fractures", but no protruding or bulging discs. Adams et al (1988) compressed and hyperextended spinal segments to the point of spinous process fractures, and no disc herniations occurred. Shea and colleagues hyperextended spinal segments to the point of spinal ligamentous and disc tears, but no disc herniations occurred. Miller et al (1986) applied shear to spinal segments in all directions and separated the disc from the vertebral body endplate, but no disc herniations occurred. Myers and colleagues applied torsion to spinal segments to the point of spinal ligamentous tears, but no disc herniations occurred. Yoganandan et al (1996) applied tension loads to spinal segments and separated the disc from the vertebral body endplate, but no disc herniations occurred. Adams and Hutton (1982) were able to create annular protrusion and nuclear extrusion, but it required posterior ligamentous or vertebral injury. They applied hyperflexion (6-18 degrees between adjacent vertebrae) and massive compression (600-3,000 lbs.). Researchers subjected small female and large male cadavers to rear end sled tests at speeds of 9 – 15 mph using a seat with no head restraint (Yoganandan 2000). Injuries were documented at all speeds except the lowest, including ligament and disc tears, facet joint widening and vertebral fractures. No disc protrusions, bulges, or herniations were seen.

Although disc protrusions and herniations cannot be generated traumatically under realistic loading conditions in cadaveric specimens, they can reliably be generated by repeated cyclic loading at physiological levels of force (Adams 1985, Wilder, Gordon, Callaghan, Tampier). In these studies, disc protrusions developed gradually and in some cases the nucleus pulposus eventually extruded through a degenerated annulus (annular fissure). The failures typically occurred progressively over thousands of loading cycles, and not as sudden events. Disc

degeneration usually begins in the second or third decade of life (Fischgrund, Garfin). However, Kjaer and colleagues found signs of lumbar disc degeneration on MRI in approximately one-third of 13 year olds studied. Tertti and co-workers had similar results: 32% of 15 year olds studied had lumbar degenerative changes. Spinal degeneration is common among asymptomatic individuals. More than 25% of asymptomatic people less than 40 years of age and almost 60% of asymptomatic people older than 40 years of age have cervical degenerative disc disease that can be identified on MRI (Boden). Matsumoto and co-workers found that nearly 40% of asymptomatic individuals in their 40s had cervical disc degeneration.

Mr. Stiebens also had a bilateral pars defect and spondylolisthesis. Spondylolisthesis is categorized by type (Shah): congenital, lytic, degenerative, traumatic, and pathologic. Genetics plays a role in the susceptibility of an individual to developing congenital or lytic types. Certain activities (gymnastics, diving, weightlifting and volleyball) are also risk factors for the development of spondylolisthesis. These activities involve hyperextension which results in increased shear stresses in the pars interarticularis. This has been theorized to lead to fatigue fractures of the pars. Degenerative spondylolisthesis is common in older people, affecting about 30% of females, and 12% of males (Kanter). It is the result of years of disc and facet degeneration, causing alteration of spinal alignment and dysfunction of load management. Jarvik and colleagues studied asymptomatic people over three years, and found some with new spondylolisthesis, some with no change in their previous spondylolisthesis, and some with resolution of the spondylolisthesis. Feffer noted the common occurrence of slippage in women over 40 between the 4^{th} and 5^{th} lumbar vertebrae, with an average movement of 2 mm every four years. Traumatic spondylolisthesis is caused by an acute fracture of the pars interarticularis (White). Mr. Stiebens' spondylolisthesis was either Type I or II and was not caused by the subject event. In fact, the pars defects and spondylolisthesis were visible on pre-incident lumbar x-rays. Recurrent episodes of back pain are common in individuals with spondylolisthesis and are almost always treated conservatively.

The notion that an asymptomatic condition can be rendered symptomatic from a one-time loading event is not supported by the literature. There would have to be a change in the spinal structure which would be demonstrated on imaging. Mr. and Mrs. Stiebens' imaging studies did not demonstrate any evidence of acute injury. Furthermore, Castro (1997), Hong, Park and Szabo (1994) performed 73 impacts between 3 and 10 mph on male and female volunteers aged 28-58 with varying degrees of degenerative changes. Volunteers with and without degenerative changes complained of mild, transient symptoms after the tests. Subjects underwent MRI before and after the collisions. There were no significant differences in MRI findings up to three months after testing.

Mr. Stiebens' records indicated that he was unaware of the impending impact. Mrs. Stiebens' records were inconsistent. Regardless, vehicle occupants unaware of an impending impact have been found to be at a higher risk of muscular symptoms, but not a higher risk of lasting injury (Anderson, Ivory).

The subject impact would certainly have been expected to cause some stress, and likely muscular symptoms. However, the symptoms would be transient and self-limiting. Castro and colleagues (2001) studied the effects of stress associated with placebo collisions. Almost 20% of the subjects experienced physical symptoms including neck and back pain, shoulder pain, vomiting, headache, dizziness, poor concentration, and numbness, despite having been involved in only a placebo collision.

The lay public, and some physicians, attribute cause to an incident when symptoms were not present prior to the incident, but were present after the incident. The American Medical Association (AMA) has published guides to be used when assessing causality (Melhorn). "A direct causal association exists only when the event is necessary and sufficient for the outcome to have occurred." The *AMA Guides* goes on to state, "there is insufficient evidence to attribute the cause of lumbar disc herniation to any minor trauma event" (Eskay-Auerbach). In the *Guides to the Evaluation of Permanent Impairment*, Rondinelli states, "causality is an association between a given cause (an event capable of producing an effect) and an effect (a condition that can result from a specific cause) with a reasonable degree of medical certainty." In the subject incident, the event was not reasonably capable of producing the effect of degenerative changes in Mr. and Mrs. Stiebens' spinal discs, as these are the result of repetitive stress over a period of years. Additionally, Mr. Stiebens did have on-going neck and back pain prior to the subject incident, and was seen for the same one month prior to the subject collision. Regarding the potential for head injury, the subject impact was not reasonably capable of producing the effect of a traumatic brain injury, even an mTBI, as the accelerations the Stiebenses experienced were well below those expected to cause injury.

Conclusions

- The subject impact was minor, and would not be expected to cause any injury.
- While both Mr. and Mrs. Stiebens reported neurologic-type symptoms, neither underwent brain imaging or neurologic evaluation to indicate the presence of a brain injury.
- The Stiebenses' recall of the incident, and their presentation to medical providers near the time of the incident were inconsistent with traumatic brain injury, even a concussion or mTBI.
- Mr. Stiebens' neck, back, and extremity pain, as well as extremity tingling predated the subject incident, and were, therefore, not caused by the collision.
- Similarly, his pars defects and spondylolisthesis predated the incident, and were, therefore, not caused by the collision.
- Mr. and Mrs. Stiebens' imaging studies demonstrated degenerative changes in their spines. Those changes were the result of repetitive stress over a period of years, and are not the result of any one time event.

As additional information is made available to me or as new facts are uncovered during the investigation and discovery process, my professional opinions may change to reflect the newfound information. The opinions expressed herein, to a reasonable degree of medical and scientific certainty, however, are current and accurately reflect my conclusions based upon the information reviewed and the analysis performed as of this date.

Should you require additional information, please do not hesitate to contact me.

Sincerely,

Lisa P. Gwin, D.O., B.S.E.E.

LPG/rss
Attachments: Bibliography
Curriculum Vitae
Testifying History
Rate Schedule
Dr. C. Day's Imaging Report
Exhibit List

Exhibit List

- Illustrations depicting vehicle dynamics
- Illustrations depicting occupant kinematics scenarios
- Illustrations depicting specific and general principles of physics
- Illustrations depicting specific and general principles of biomechanics
- Illustrations depicting specific and general principles of injury causation
- Illustrations depicting relevant injury mechanisms
- Illustrations depicting relevant degenerative mechanisms
- Timeline of medical history
- Selected imaging studies
- Selected medical records
- Selected vehicle photographs
- Diagram of impact sequence
- Selected exemplar vehicle/surrogate photographs
- Data/graphs related to vehicle/occupant dynamics and injury potential
- Relevant data and graphs
- Excerpts from relevant/related medical and biomechanical literature